

What is claimed:

1. An isolated nucleic acid molecule having a sequence selected from the group consisting of:
  - (a) SEQ ID NO:1;
  - (b) a nucleotide sequence that hybridizes under stringent conditions to the complement of the nucleotide sequence of (a) and which encodes MINC102, wherein the stringent conditions are 30% formamide in 5 x SSPE (0.18 M NaCl, 0.01 M NaPO<sub>4</sub>, pH 7.7, 0.001 M EDTA) buffer at a temperature of 42°C and remaining bound when subject to washing at 42°C with 0.2 x SSPE; or
  - (c) a nucleotide sequence which, as a result of the degeneracy of the genetic code, differs from the nucleic acid of (a) or (b) and which encodes MINC102.
2. A human MINC102 protein encoded by the nucleic acid sequence of claim 1.
3. A human MINC102 protein comprising SEQ ID NO:2, or biologically active fragments thereof.
4. A method for identifying an agent capable of inhibiting the expression or activity of MINC102, comprising:
  - (a) administering a test agent to an animal expressing a MINC102 protein, or protein fragment; and
  - (b) determining the ability of the test agent to inhibit MINC102 expression or activity.
5. The method of claim 4, wherein the MINC102 protein comprises the amino acid sequence of SEQ ID NO:2, or a fragment thereof.
6. A therapeutic method for treating, inhibiting, or reducing muscle atrophy in a subject in need thereof, comprising administering a therapeutically effective amount of an agent capable of inhibiting MINC102 activity or expression.
7. The therapeutic method of claim 6, wherein the agent is capable of inhibiting MINC102 activity.
8. The therapeutic method of claim 7, wherein the agent is an antagonist of MINC102.
9. The therapeutic method of claim 8, wherein the antagonist is an antibody to MINC102.
10. The therapeutic method of claim 9, wherein the antibody may be polyclonal, monoclonal, chimeric, humanized, or a wholly human antibody.
11. The therapeutic method of claim 8, wherein the antagonist is an activator of the Akt pathway.

12. The therapeutic method of claim 11, wherein the antagonist is insulin-like growth factor 1 (IGF-1), clenbuterol, albuterol, or salbuterol.
13. The therapeutic method of claim 6, wherein the agent is capable of inhibiting MINC102 expression.
14. The therapeutic method of claim 13, wherein the agent is an antisense molecule, a ribozyme or triple helix, or a short interfering RNA (siRNA) capable of silencing MINC102 gene expression.
15. An isolated nucleic acid sequence encoding a mouse MINC102, comprising:
  - (a) SEQ ID NO:3; or
  - (b) a nucleotide sequence which, but for the degeneracy of the genetic code, would hybridize to the complement of SEQ ID NO:3, and which encodes a molecule having the biological activity of MINC102.
16. A transgenic animal, comprising a modification of an endogenous MINC102 gene.
17. An antibody which specifically binds the MINC102 protein of claim 3.